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Four runners (R's; $\dot{V}O_2max$ 64.9 $\pm$ 2.7 ml/kg.min, $\bar{x} \pm$ S.D.) and six nonrunners (NR's; $\dot{V}O_2max$ 52.0 $\pm$ 5.4 ml/kg.min, $\bar{x} \pm$ S.D.) were studied during various exercise tests in order to compare venous lactate accumulation. Subjects were studied on three occasions while walking on a treadmill with increases in grade at 1, 3 and 5 minute intervals. Based upon the results of these tests, each subject performed for 30 minutes, at an individually selected workload which was just above the level at which venous lactate began to increase exponentially. During the incremental work tests, lactate accumulation was delayed, both in

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terms of relative and absolute workload, in trained R's. Lactate accumulation was altered by the different time intervals during the incremental tests. During the 30 minute walk, both R's and NR's showed similar lactate accumulation during the first five minutes of exercise. Between 5 and 10 minutes, R's had a mean decrease of 0.33 $\pm$ 0.44 mM/L ($\bar{x} \pm$ S.D.) while NR's had a mean increase of 0.63 $\pm$ 0.73 mM/L ($\bar{x} \pm$ S.D.) ($p < 0.05$). This suggests that the accumulation of venous lactate is reversible during steady state exercise in trained individuals.

Lactate Accumulation for Runners and Non-Runners During Various Exercise Tests

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Running Head: Lactate in Runners and Non-Runners.

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Abstract

Four runners (R's; $\dot{V}O_2$ max 64.9 ± 2.7 ml/kg • min, $\bar{x} \pm$ S.D.) and six non-runners (NR's; $\dot{V}O_2$ max 52.0 ± 5.4 ml/kg • min, $\bar{x} \pm$ S.D.) were studied during various exercise tests in order to compare venous lactate accumulation. Subjects were studied on three occasions while walking on a treadmill with increases in grade at 1, 3 and 5 minute intervals. Based upon the results of these tests, each subject performed for 30 minutes, at an individually selected workload which was just above the level at which venous lactate began to increase exponentially. During the incremental work tests, lactate accumulation was delayed, both in terms of relative and absolute workload, in trained R's. Lactate accumulation was altered by the different time intervals during the incremental tests. During the 30 minute walk, both R's and NR's showed similar lactate accumulation during the first five minutes of exercise. Between 5 and 10 minutes, R's had a mean decrease of 0.33 ± 0.44 mM/L ($\bar{x} \pm$ S.D.) while NR's had a mean increase of 0.63 ± 0.73 mM/L ($\bar{x} \pm$ S.D.) ($p < 0.05$). This suggests that the accumulation of venous lactate is reversible during steady state exercise in trained individuals.

Blood lactate concentrations have been measured for many years as an indicator of the contribution of anaerobic processes to energy production (1, 2, 6, 7, 10-12, 14, 15, 30). It has been demonstrated that, during incremented exercise, an individual reaches a point at which lactate begins to increase exponentially in the blood. Anaerobic threshold (AT) and lactate turning point (LTP) are terms used to describe the point just below the onset of the exponential increase in lactate (5, 22, 25, 27, 28). Many of the studies measured AT or LTP from ventilatory changes during incremental exercise on a cycle ergometer (25, 27-29). Since very little has been reported on treadmill exercise, one purpose of this study was to compare treadmill walking tests in which the exercise load was incremented at various time intervals in order to compare different protocols for determining LTP while measuring venous lactate. Previous studies have also shown that at the same absolute workload trained individuals have less lactate accumulation than untrained subjects (8, 13). It has also been demonstrated that even at the same relative (% $\dot{V}O_2$ max) workload that lactate production is less in trained versus untrained individuals (8, 16). Therefore, lactate accumulation appears to be dependent on several factors to include absolute and relative workload and state of training. However, previous studies which have compared lactate levels at submaximal workloads in trained and untrained subjects have not taken LTP into account. Therefore, a second purpose for this study was to compare lactate accumulation in trained and untrained subjects at a workload at which lactate has been demonstrated to begin to accumulate in venous blood.

METHODS

Ten male soldiers volunteered to participate in this study. Four were runners (Rs) who trained regularly. Three of these runners completed a marathon within a three month period prior to the start of the study with times

of 2 hrs, 44 minutes, 3 hrs even, and 3 hrs, 34 minutes. The other six subjects were young soldiers who were physically active in recreational sports (e.g., basketball, softball, weightlifting) but did not participate in any regular training program. Before participation, informed consent was obtained and subjects were familiarized with the test procedures. Subjects underwent a series of tests which included the following:

(1) Anthropometric measures (height and weight),

(2) Running maximal oxygen uptake ($\dot{V}O_2$ max R),

(3) One-minute incremental walking test,

(4) Three-minute incremental walking test,

(5) Five-minute incremental walking test, and

(6) Thirty-minute walk at a single sub-maximal workload.

A running $\dot{V}O_2$ test was the initial procedure performed by each subject. This was a modification of the interrupted treadmill test described by Mitchell et al (18). An initial run for 6 minutes at 161 m/min (6 mph) 0% grade was followed by a 5-10 minute rest period. Two to four additional runs of 3-4 minutes were performed, each followed by a rest period. Workloads were increased by adjusting speed and/or grade. During the last minute at each work load, expired air was collected in Douglas bags. Subjects breathed through a mouthpiece attached to a Koegel y-valve. A plateau in oxygen consumption (increase $<$ 2 ml/kg•min per 2.5% grade increase) was defined as $\dot{V}O_2$ max R. Expired air was analyzed with a Beckman LB-2 CO_2 analyzer and an Applied Electrochemistry, Inc. S3-A O_2 analyzer.

On subsequent days, following the $\dot{V}O_2$ max run, test subjects performed a series of treadmill protocol walking tests. The first session consisted of walking at 80.5 m/min (3 mph) with a 3% grade increase each minute until the subject

could no longer continue. During the last 30 seconds at each workload, expired air was collected and $\dot{V}O_2$ determined. In addition, a one ml sample of venous blood was withdrawn for lactate determination. Blood was withdrawn through a 19 gauge prediatric needle, which was inserted into a vein on the back of the hand or in the forearm. Drawn blood samples were immediately placed in 8% perchloric acid and later analyzed by an enzymatic kit (Sigma Chemical Co., St. Louis, MO).

The next procedure consisted of walking as described above except that the workload was increased every three minutes until the subject could no longer continue. $\dot{V}O_2$ was measured as above, and blood lactate was measured after 1 minute and 3 minutes at each workload. Maximal oxygen consumption for walking ($\dot{V}O_2$ max W) was determined from the results of the one and three-minute incremental tests.

For the next session, subjects walked for five minutes at five workloads. Workloads were selected for each subject so that two were below the point where venous lactate began to accumulate exponentially, one was at or near that point and two were above it. $\dot{V}O_2$ was measured at the end of each workload and lactate after 1,3 and 5 minutes at each workload.

In the final procedure, subjects walked for 30 minutes at a single work load. Based upon the results of the previous walks, a workload was selected for each subject which was the lowest load at which venous lactate began to increase exponentially in any of the three previous tests. All subjects walked for three minutes at 80.5 m/min (3 mph) 0% grade and then went immediately to the appropriate grade and walked for 30 minutes. Samples for lactate determination were withdrawn at 1,3,5,8,10,13,15,18,20,23,25,28, and 30 minutes. $\dot{V}O_2$ was measured every five minutes.

All walking tests were performed at 80.5 m/min (3 mph) and at least one day of rest separated procedures. No more than two procedures were performed in any one week. Statistical comparisons were made using an analysis for variance for repeated mesures or an unpaired t-test. Tukey's Test was used for post-hoc comparisons. For statistical significance, we used $p \leq 0.05$.

RESULTS

Table 1 lists the basic physical data and the running $\dot{V}O_2$ max for each subject. The running $\dot{V}O_2$ max of Rs was 23% higher ($p < 0.005$) than in non-runners (NRs).

Table 2 summarizes the physiological data collected during the maximal oxygen uptake tests while running ($\dot{V}O_2$ max R) and walking ($\dot{V}O_2$ max W). $\dot{V}O_2$ max W was 10.8% and 19.7% lower than $\dot{V}O_2$ max R in NRs and Rs, respectively. HR and $\dot{V}_E$ at $\dot{V}O_2$ max were also lower for walking than running.

Figures 1, 2, 3 illustrate the results of venous lactate determinations while walking with grade increases at 1, 3 and 5 minute intervals, respectively. In Figure 1, Rs showed a later onset of lactate accumulation, as a function of absolute grade, compared to NRs. Similar results are in shown Figure 2, which illustrates venous lactate levels during the 3 minute incremental walking test. However, the lactate curve shifted noticeably to the left in NRs and slightly to the left in Rs. This indicates that the lactate turning point (LTP) was reached at a lower absolute workload (% grade) when the time at each workload was increased. Table 3 lists the individual grades at which each subject exceeded the LTP for the various exercise tests. Listed are the workloads at which lactate showed the initial sustained increase above the baseline values for each subject. Figure 3 illustrates venous lactate values for Rs and NRs during the 5 minute

incremental treadmill walk. The figure illustrates that the first two workloads were below the LTP, the third was at or just above the LTP and the last two were above the LTP. Individual inspection of the data showed that 4 of the 6 NRs had definite lactate accumulation after 5 minutes at 12% grade and all showed substantial accumulation after 5 minutes at 18% grade. For R's, two began to accumulate lactate after 5 minutes at 18% with definite accumulation at 24% grade. The other two showed some accumulation after 5 minutes at 24% grade and definitely after beginning to walk at 30% grade. Again, we see a lowering of the absolute workload at which the LTP occurs with a longer duration at each workload, but only in trained subjects.

Based upon the results of these treadmill tests, a workload just above the LTP, was individually selected for the thirty minute walk (Figure 4). The mean workload was 14% grade (range 12-15%) and 23.8% (range 18-27%) for NRs and Rs, respectively. The increase in venous lactate was virtually identical for the first five minutes of the walk in both groups. After five minutes, however, NRs continued to climb to a plateau which was maintained throughout the duration of the walk. Rs, on the other hand, had a decrease in venous lactate which plateaued at a lower level than NRs. Between 5 and 10 minutes, Rs had a mean decrease of 0.33 ± 0.44 mM/L ($\bar{x} \pm$ S.D.), while NRs had a mean increase of 0.63 ± 0.73 mM/L ($\bar{x} \pm$ S.D.). This difference was significant at the .05 level. Minute ventilation, $\dot{V}O_2$ and $\dot{V}_{CO2}$ were higher in Rs than in NRs which is consistent with the fact that there were differences in workloads. Heart rate, respiratory quotient (R) and ventilatory equivalent (VEQ) are approximately equal in both groups. Both groups were working at approximately the same % of $\dot{V}O_2$ max R at 15 minutes into exercise, 60.6% and 64.8% for NRs and Rs respectively.

DISCUSSION

The major finding of this study suggests that the accumulation of venous lactate is reversible during steady state exercise above the LTP in trained individuals. While there is general agreement that training delays the onset of lactate accumulation in man, there are conflicting reports as to the effect of prolonged exercise on lactate. In 1936, Bang (2) reported an initial rise, followed by a steady fall, in the blood lactate concentration during constant exercise of long duration. This was the typical response that occured at several exercise intensities. Other investigators (20, 21) have reported results which agree with Bang. However, reports have also been published in which this fall in lactate has failed to occur (3, 27). In a study by Cobb and Johnson (3), physically active men showed the initial rise and fall while sedentary men showed a steady increase in lactate. However, in this study, both groups worked at the same workload. Therefore, differences in the workloads at which lactate accumulation begins to occur were not taken into account. In another study, Wasserman et al (26) reported on the effect of prolonged exercise (50 minutes) on blood lactate levels in individuals working at various workloads. Workloads were assigned based upon a previous assessment of the subject's anaerobic threshold, however, there was no indication of the individual's level of fitness. Subjects demonstrated a varied lactate response to prolonged exercise.

In the present study subjects were divided into two distinct groups based upon training activity and thus level of fitness. While all subjects were healthy and relatively fit, there was a significantly higher level of fitness in the trained runners. When a comparison is made of the lactate response to prolonged exercise, we see that there is a very distinct response for both groups. Runners showed the initial rise which was followed by a decrease in the lactate level,

similar to reports by Bang (2). This type of response was not seen in the non-runners. The response of runners and non-runners was virtually identical for the first five minutes of exercise. It was after this period of time that the differences began to appear. Non-runners continued to increase lactate until a plateau was achieved after 8-10 minutes. Runners peaked at 5 minutes of exercise and displayed a steady fall in lactate until 18 minutes of exercise. At 30 minutes there is a slight increase in the lactate level of runners similar to the secondary rise reported by Bang (2). It is stated by Bang that his subjects "were well trained in these and similar types of exercise, and this condition was maintained throughout the period of investigation". Cobb and Johnson (3) obtained similar results in their physically active subjects.

The results of the present study indicate that training alters the accumulation of venous lactate. There are several factors that could account for this. There are several cellular adaptations which take place with endurance training which could account for this difference. Trained individuals have a greater reliance on fat oxidation as a source of energy as opposed to carbohydrate (10). The initial rise in lactate may be due to production which occurs in the muscle cells prior to mobilization of fatty acids. Once free fatty acids are mobilized, trained subjects could conceivably demonstrate a reduction in lactate because of the preferential shift to fats as an energy source.

It has also been reported that endurance trained rats have an increase in alanine transaminase activity (10). This could cause an increased conversion of pyruvate to alanine and less to lactate. However, Hermansen and Vaage (9) reported that this reaction was of minor importance in terms of lactate removal in healthy young men after maximal exercise. There is no mention of the level of fitness in these individuals and it may be that this reaction is only enhanced

after endurance training. Hermansen and Vaage report that the majority of lactate produced is most probably converted back to glycogen. Such a mechanism could explain the decrease in venous lactate seen in the trained runners if little or no additional lactate was being produced after five minutes because of a shift to fatty acids as an energy source. However, this reaction has only been demonstrated to occur after exercise. Whether or not it is proceeding during exercise and whether or not it is enhanced by endurance training is not known.

Another possible cause for the decrease in lactate which occurred in R's may be a difference in the activity and distribution of the LDH (lactate dehydrogenase) isozymes between the two groups. Sjodin (23) reported with endurance training that total LDH activity decreased, and the relative contribution of H-LDH, and the H/M ration increased. H-LDH increases as the % of slow twitch muscle fibers increases (23). H-LDH catalyzes the conversion of lactate to pyruvate while M-LDH catalyzes the reverse reaction. While no muscle biopsies were taken during this study, it is possible that the difference in lactate response to prolonged work may be due to differences in enzyme activities brought about by endurance training.

Senay and Kok (22) have also reported a difference between trained and untrained subjects with regard to fluid shifts during prolonged exercise. They reported that after training subjects added fluid to the vascular compartment whereas before training fluid was lost from the vascular system. This shift occurred during a 4 hour work test and whether it is of importance in a test of lesser duration, such as in this study, is questionable. However, it does point out another difference between trained and untrained individuals which could affect the concentrations of metabolites in venous blood.

It does not appear that the lower lactate levels in the runners are due to an improvement in delivery of O_2 to working muscles, since the lactate response is identical to that of non-runners for the first five minutes of exercise. Our results support the statement by Hollozy and Booth (10) that changes in skeletal muscle, rather than improved O_2 delivery account for lower lactate levels.

Regardless of the mechanisms involved, the present study illustrates that during steady state exercise at a workload at which lactate begins to accumulate in venous blood, trained runners make adjustments which resulted in a reduction in peak lactate levels. This indicates that local tissue hypoxia is not a factor and terms which refer to an onset of "anaerobic" metabolism may be misnomers.

The trained individuals in this study had $\dot{V}O_2$ max R values similar to those reported by Costill et al. (4) in trained distance runners. Values for NRs are comparable to those previously reported for young, military personnel (19). These differences between walking and running are somewhat larger than those reported by previous authors (17, 24). This may be due to the type of walking protocols used to measure work performance in this study. McArdle et al (17) reported that subjects complained of local muscle discomfort at high treadmill elevations at 3.4 mph. Similar experiences occured in this study with a speed of 3.0 mph and very high treadmill elevations. Based upon peak heart rates achieved, it appears that local muscle fatigue and discomfort limited walking performance rather than central cardiovascular limitations. All subjects complained of discomfort in calf muscle at higher elevations. However, walking at this speed enabled us to demonstrate that subjects, especially trained runners, were able to tolerate high workloads without any significant venous lactate accumulation. It also enabled us to obtain good baseline lactate values for all subjects. If we use a blood lactate concentration of 2.2 mM/L as an indicator of

substantial lactate accumulation (16), runners reached this concentration at 33, 27, and 24 per cent grade for the 1,3, and 5 minute incremental tests, respectively (see Figures 1, 2, 3). Non-runners reached this level at 21, 15% and 18% for the 1, 3, and 5 minute tests, respectively. However, in the 5 minute test, non-runners jumped from 12 to 18% and after 3 minutes were above 2.2 mM/L. It is apparent that lactate levels are dependent not only upon the elevation at which individuals are working but also the duration of time spent at each level. It is also apparent that the absolute workload required to cause lactate accumulation is higher in the trained runners than in untrained individuals. These results support results previously reported by other investigators (8, 13, 16) that training delays the onset of lactate accumulation during incremental work. These results also indicate that the duration at each workload must be considered when determining LTP. Based upon the results of this study, a time between 3 and 5 minutes per workload seems appropriate.

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TABLE 1. Physical Characteristics of Subjects.

Subject	Ht(cm)	Wt(kg)	Age(yrs)	Running $\dot{V}_{O_2}$ max (ml/kg•min)
Non-Runners				
S1	160.8	67.1	20	57.1
S2	173.9	56.4	24	53.5
S3	168.5	57.1	27	53.3
S4	178.4	74.1	21	51.0
S5	169.2	62.1	23	47.4
S6	171.1	65.8	21	53.3
$\bar{X}$	170.3	63.8	22.6	52.6
S.D.	$\pm$5.9	$\pm$6.7	$\pm$2.5	$\pm$3.2
Runners				
S7	177.8	70.7	31	64.5
S8	181.0	68.7	30	67.9
S9	169.3	74.4	25	61.4
S10	177.0	79.1	31	65.6
$\bar{X}$	176.3	73.2	29.3	64.9
S.D.	$\pm$5.0	$\pm$4.6	$\pm$4.6	$\pm$2.7

TABLE 2. Physiological Data Collected During Maximal Walking and Running

	$\dot{V}O_2$ max (l/min)	$\dot{V}O_2$ max (ml/kg•min)	$\dot{V}_E$ BTPS (l/min)	Heart Rate (beats/min)	R
Non-Runners					
Running					
$\bar{X}$	3.34	52.6	117.4	195	1.12
S.D.	$\pm$0.39	$\pm$3.2	$\pm$14.6	$\pm$12	$\pm$.06
Walking					
$\bar{X}$	3.00	46.9	106.1	188	1.13
S.D.	$\pm$0.36	$\pm$3.5	$\pm$31.2	$\pm$9.7	$\pm$.04
Runners					
Running					
$\bar{X}$	4.65	64.9	166.2	183	1.12
S.D.	$\pm$0.23	$\pm$2.7	$\pm$14.9	$\pm$2	$\pm$.04
Walking					
$\bar{X}$	3.77	52.0	126.8	176	1.04
S.D.	$\pm$.75	$\pm$5.4	$\pm$24.6	$\pm$10	$\pm$.08

TABLE 3. Workload and % $\dot{V}O_2$ max R at Which Each Subject Began to Accumulate Lactate During the Incremental Tests and Workload Selected for the 30 Minute Walk.

	1'		3'		5'		30'
	% grade	% $\dot{V}O_2$ max R	% grade	% $\dot{V}O_2$ max R	% grade	% $\dot{V}O_2$ max R	% grade
Non-Runners							
S1	18	59.0	15	52.8	12	51.5	15
S2	15	44.1	15	51.2	12	57.6	14
S3	18	53.1	12	52.3	18	66.8	14
S4	18	69.9	12	50.2	12	54.3	15
S5	24	78.6	12	65.9	12	59.8	12
S6	18	55.6	12	59.6	12	53.8	14
$\bar{X}$	18.5	60.0	13.0	55.3	13.0	57.3	14.0
S.D.	± 2.9	± 12.4	± 1.6	± 6.2	± 2.4	± 5.5	± 1.4
Runners							
S7	27	57.7	30	69.7	24	71.2	26
S8	30	79.3	24	63.5	24	63.6	27
S9	21	55.9	21	62.0	18	53.6	18
S10	27	65.7	21	66.0	24	62.1	24
$\bar{X}$	26.3	64.7	24.0	65.3	22.5	62.6	23.8
S.D.	± 3.8	± 10.7	± 4.2	± 3.3	± 3.0	± 7.2	± 4.0

Legends to Figures

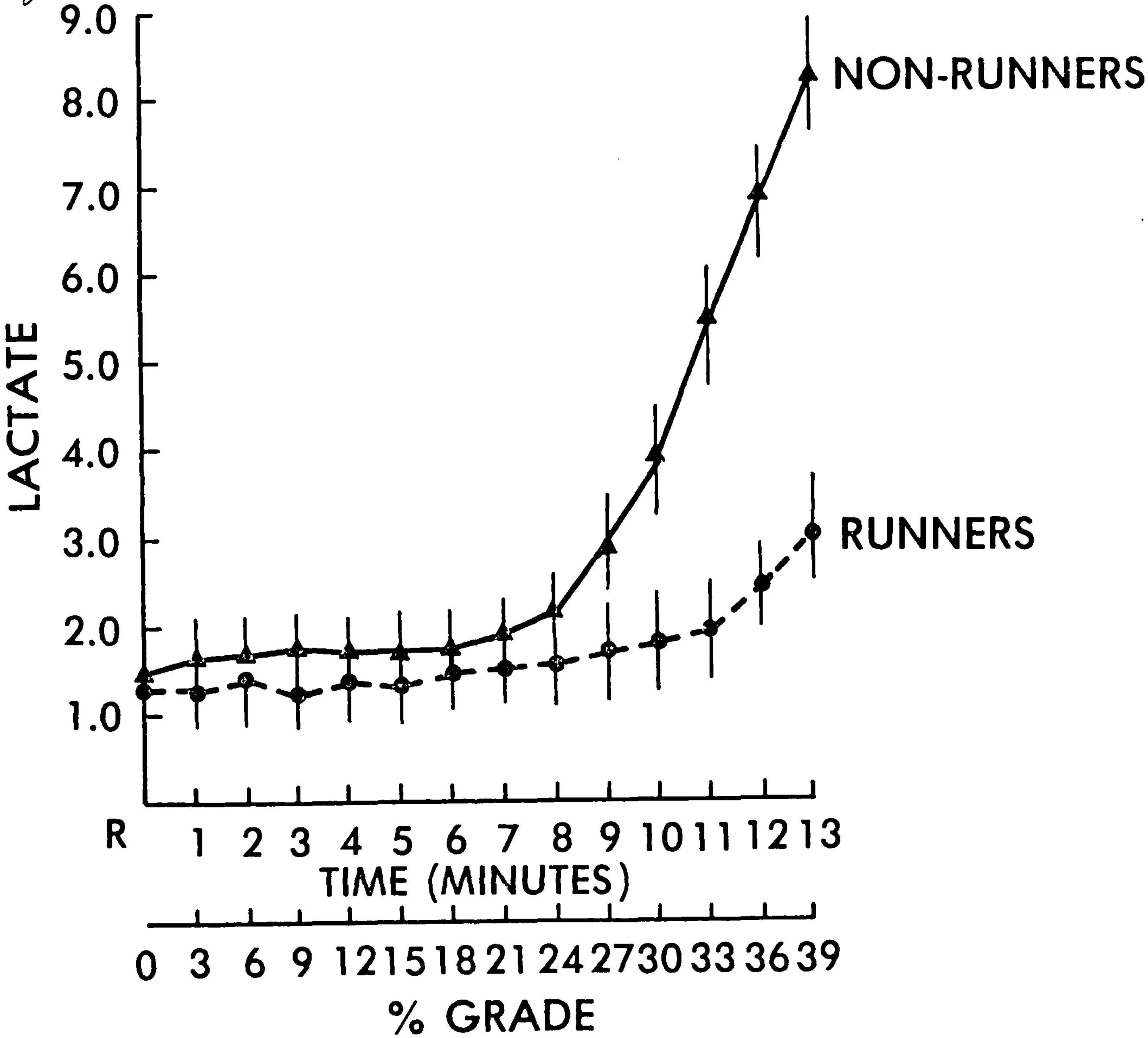
Fig. 1. Venous lactate accumulation during the 1 minute incremental walking test. For this test, subjects walked at 80.5 m/min (3 mph) with a 3% grade increase every minute until they could no longer continue. Lactates are reported as mM/L. Values expressed as $\bar{x} = \pm$ S.E.

Fig. 2. Venous lactate accumulation during the 3 minute incremental walking test. For this test, subjects walked at 80.5 m/min (3 mph) with a 3% grade increase every 3 minutes until exhaustion. The percent grade at which subjects were working corresponds to the time in minutes designated in the figure.

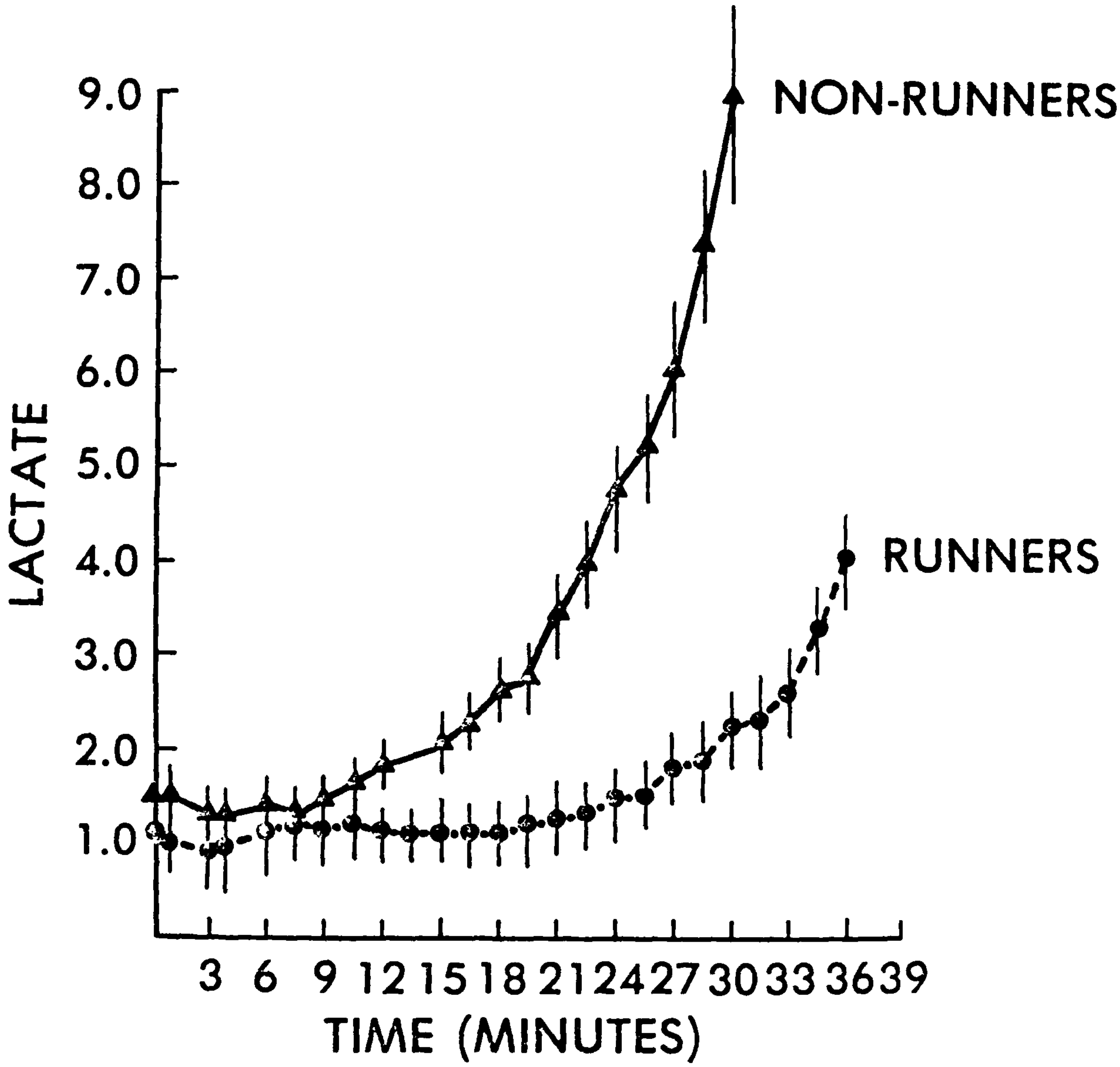
Fig. 3. Venous lactate accumulation during the 5 minute incremental walking test. For this test, subjects walked at 80.5 m/min (3 mph) for 5 minutes at 5 separate workloads. Workloads were selected so that two were below, two above and one at or near the level at which lactate began to accumulate. There is no point at 25 minutes for R's because of difficulties with the blood collected catheter in two subjects. NR's (▲——▲); R's (●--●) and % grade (——).

Fig. 4. Venous lactate accumulation and other data collected during thirty minute walk at individually selected workload. The mean grade at which R's and NR's were working is indicated.

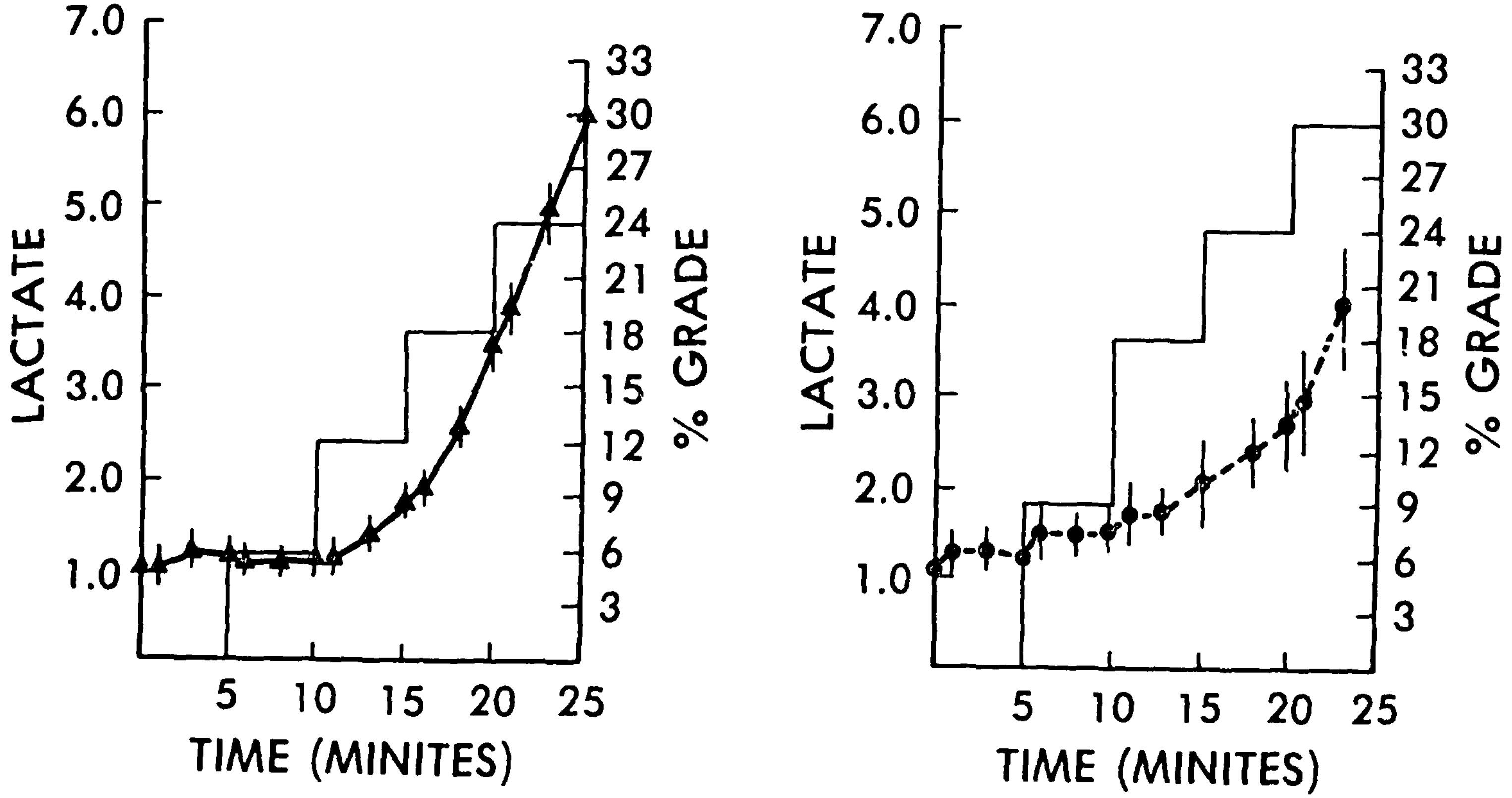
Daniel, Fig, 1



9.0
8.0
7.0
6.0
5.0
4.0
3.0
2.0
1.0
LACTATE
NON-RUNNERS
RUNNERS
R 1 2 3 4 5 6 7 8 9 10 11 12 13
TIME (MINUTES)
0 3 6 9 12 15 18 21 24 27 30 33 36 39
% GRADE

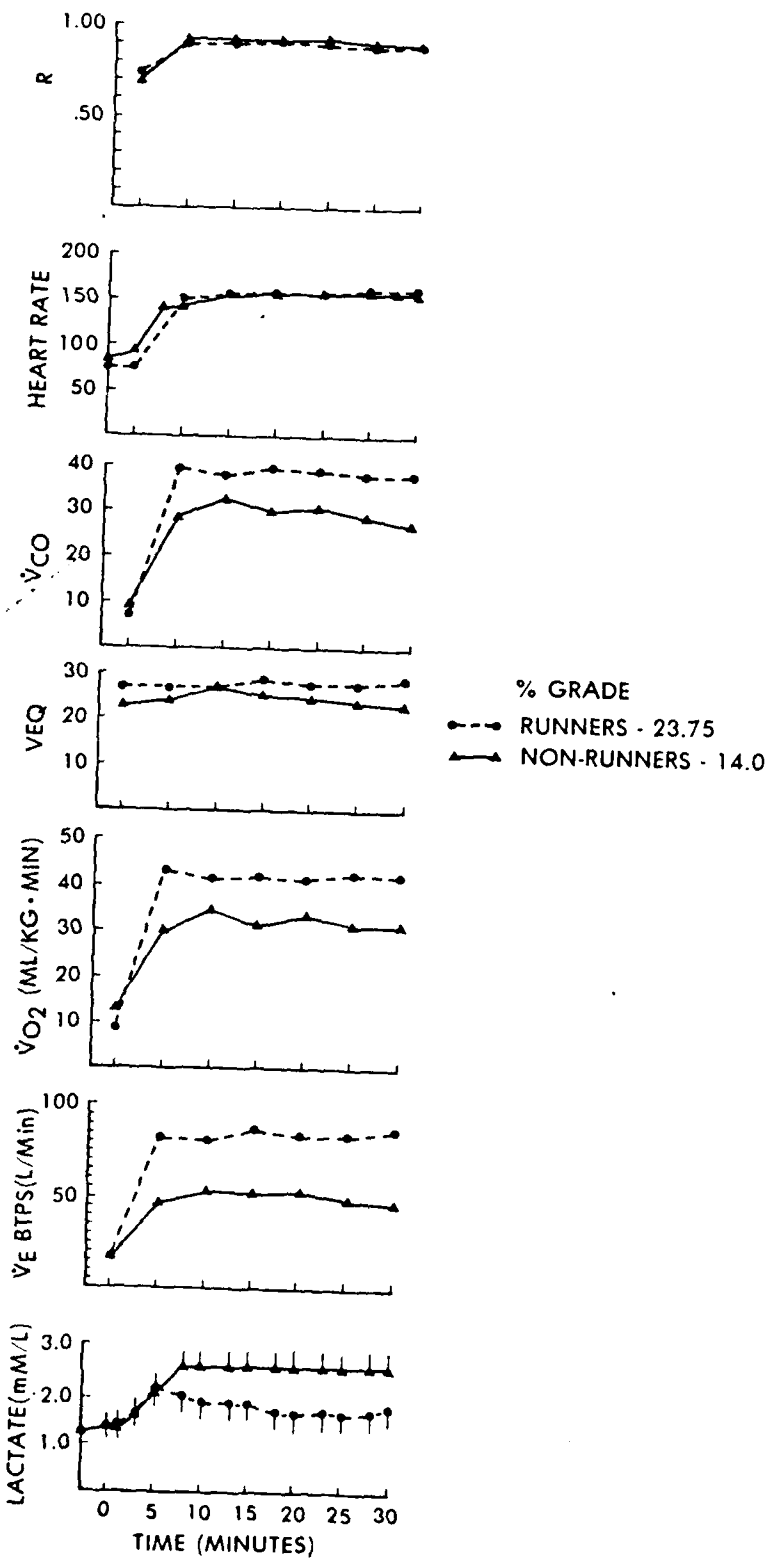


Daniels Fig - 2


LACTATE
7.0
6.0
5.0
4.0
3.0
2.0
1.0
33
30
27
24
21
18
15
12
9
6
3
% GRADE
5 10 15 20 25
TIME (MINITES)
LACTATE
7.0
6.0
5.0
4.0
3.0
2.0
1.0
33
30
27
24
21
18
15
12
9
6
3
% GRADE
5 10 15 20 25
TIME (MINITES)

Daniel Fig 3

Daniels Fig. 4



The views, opinions, and/or findings contained in this report are those of the author(s) and should not be construed as an official department of the Army position, policy, or decision, unless so designated by other official documentation.

Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

END

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